INSECTICIDE RESISTANCE IN COTTON APHID LINKED TO FIELD-CONTROL FAILURES IN AUSTRALIAN COTTON

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Abstract
Australia-wide insecticide resistance surveying of cotton aphid populations identified pirimicarb, organophosphate, endosulfan and pyrethroid resistance. Resistance levels were often high to extreme and have been linked to field control failures for the first time. Resistant cotton aphids have the potential to seriously impact the Australian cotton industry and their resistance management should now be an industry priority.

Introduction
Cotton aphid, *Aphis gossypii* Glover, is the main aphid pest of cotton throughout the world causing significant problems due to honeydew contamination of the open boll lint (Schepers 1989). In Australia, cotton aphid is an important late season cotton pest and is specifically targeted for insecticidal control or is coincidentally controlled by sprays directed at other pests. Overseas studies have found cotton aphid populations that were resistant to all the major insecticide groups. In Australia, resistance in cotton aphid has already been detected but control failures have not been related to resistance (Herron *et al.* 2000).

Transgenic cotton expressing the Cry IAc gene for *Bacillus thuringiensis* (Ingard®) accounted for about 20% of the cotton area in the 1998-99 season (Fitt 1998). Typically, insecticide use is halved on transgenic cotton, mostly in the first half of the season. Fewer early season sprays may allow aphid populations to increase to economic levels earlier in the transgenic cotton. Treatments specifically targeting cotton aphid may increase selection for resistance causing honeydew contamination of the cotton lint.

This study aimed to measure the frequency and levels of pyrethroid, organophosphate, carbamate and endosulfan resistance in cotton populations from all major cotton growing districts in Australia.

Materials and Methods
Insecticides
Products, their supplier, common name, chemical group, formulation and active ingredient concentration are given in Table 1.

Aphids
Field population samples of cotton aphid were collected Australia-wide and sent by

overnight courier for testing. Aphids were reared on pesticide-free cotton, variety 'Deltapine 90', at 25±4 °C under natural light. Strain integrity was assured by maintaining populations in purpose built aphid proof cages. Strains were collected from cotton or intensive horticulture in close proximity to cotton.

Bioassay method

The method of testing *A. gossypii* is as described in Herron *et al.* (2000). Briefly, the

Table 1 Supplier, trade name, common name, chemical group, formulation and concentration for seven pesticides tested against cotton aphid

Supplier	Trade Name	Common Name	Chemical Group	F#	Conc.
Crop Care	Endosulfan	endosulfan	organochlorine	EC	350 g/L
Bayer	Folimat	omethoate	organophosphate	LC	800 g/L
Crop Care	Pirimor	pirimicarb	carbamate	WP	500 g/Kg
Novartis	Curacron	profenofos	organophosphate	EC	500 g/L
Crop Care	Talstar	bifenthrin	pyrethroid	EC	100 g/L
Cyanamid	Hallmark	esfenvalerate	pyrethroid	EC	50 g/L
AgrEvo	Decis Forte	deltamethrin	pyrethroid	EC	27.5 g/L

F# = formulation: EC=emulsifiable concentrate, LC=liquid concentrate and WP=wettable powder.

method utilised 35 mm Petri dishes into which an excised cotton plant leaf disc was placed onto 3 mL of cooling liquid agar. When the agar had set, batches about 20 adult aphids were transferred onto the leaf discs. Leaf disc and aphids were then sprayed by a Potter spray tower producing an aqueous deposit of 1.6 ± 0.07 mg cm^{-2} with a 2 mL aliquot. Discriminating concentrations (LC99.9 value for a susceptible strain) were used to determine the presence/absence of resistance (Herron *et al.* 2000). Strains with survivors at the discriminating-dose were further tested against a range (3-5) of concentrations to produce log-dose probit (l-d p) assays. These were replicated a minimum of four times and included a water only sprayed control. After spraying, Petri dishes were covered with finely perforated clear plastic film that maintained high humidity but prevented condensation. Tests were maintained at 25 ± 0.1 °C in constant light for 24 h until mortality was assessed.

Analysis

LC50 and LC99.9 values were computed on an IBM compatible PC using Probit 5 for Windows and included control mortality correction (Gillespie 1995). Resistance factors were calculated by dividing the LC50 (99.9) of the field collected strain by LC50 (99.9) of a laboratory susceptible strain.

Results

Curacron resistance was moderate to low and restricted to strains collected from Qld, WA and the NT (Table 2). Similarly, no Folimat resistance was detected in NSW, but high level resistance was found elsewhere. Extreme Pirimor resistance was measured in Qld, WA and NT but NSW populations were generally susceptible. Endosulfan resistance was low and largely restricted to NSW. Decis Forte resistance was again generally restricted to NSW, with resistance levels moderate to high. In contrast, Talstar and Hallmark resistance tended to be ubiquitous with resistance levels ranging from low to high.

Table 2. Dose response summary for strains of cotton aphid collected from major Australian cotton growing regions and tested for resistance against several pesticides

Chemical	State	Populations tested	Populations resistant	Highest LC50 level resistance	Highest LC99.9 level resistance
Curacron	NSW	10	0	-	-
	Qld	5	5	4.8	18
	WA	7	6	6.3	17
	NT	3	3	5.0	4.1
Folimat	NSW	10	0	-	-
	Qld	5	4	51	92
	WA	7	5	46	81
	NT	3	3	56	67
Pirimor	NSW	10	1	1.8	4.2
	Qld	5	5	1,727	20,044
	WA	7	5	4,909	84,066
	NT	3	3	4,016	19,648
Endosulfan	NSW	10	7	8.2	6.9
	Qld	5	1	1.2	1.1
	WA	7	0	-	-
	NT	3	0	-	-
Decis Forte	NSW	10	7	19	30
	Qld	5	0	-	-
	WA	7	1	NA	NA
	NT	3	0	-	-
Talstar	NSW	10	7	48	78
	Qld	5	1	1.3	3.4
	WA	7	4	2.7	8.3
	NT	3	0	-	-
Hallmark	NSW	10	7	27	39
	Qld	5	3	2.2	6.9
	WA	7	1	NA	NA
	NT	3	1	4.6	4.1

- = not tested, discriminating-dose indicated no resistance

NA = not available, strain killed by parasites before testing complete

Discussion

This is there first time that resistance in cotton aphid from Australian cotton has been linked directly with field-control failures. Control failures were reported in all Emerald strains, and one strain could only be controlled with Pegasus® (diafenthiuron). Resistant cotton aphids have the potential to seriously impact the Australian cotton industry and their resistance management should now be an industry priority.

Endosulfan resistance in cotton aphid was negligible or not detected in Queensland populations associated with control failure (Table 2). Clearly, endosulfan has potential for use against resistant populations but its use is restricted to the earlier first and second stages of the season as part of the cotton bollworm resistance management strategy (Harris & Shaw 1998). For the current 1999 / 2000 cotton season, endosulfan use has been further restricted to a maximum of three full coverage applications (QDPI 1999). We consider that the progressive reduction in endosulfan use in Australian cotton may be contributing to the increasing instances of cotton aphid requiring specific aphicide control.

There is a general dichotomy of response between strains collected from NSW and elsewhere in Australia. We think these differences relate to overall insecticide use. As resistance likely relates to spray intensity, we suggest that the ubiquitous distribution of esfenvalerate and bifenthrin resistance in cotton aphid indicates a general overuse of these products for cotton aphid control in Australia.

We detected pyrethroid resistance particularly in NSW populations. Cotton aphid probably endures high pyrethroid selection as there is high usage of this group against the major cotton pests (*Helicoverpa* spp.). Currently, pyrethroids are not recommended for cotton aphid control and given the level of pyrethroid resistance and the restrictions placed on their use (Harris & Shaw 1998) it is unlikely they will be useful.

The organophosphate and Pirimor resistances detected in this study (Table 2) are similar to levels reported for overseas populations. Moores *et al.* (1996) and Delorme *et al.* (1997) identified insensitive acetylcholinesterase (AChE) as the major mode of action for resistance with little detoxification evident. As the major underlying mechanism to resistance in Australian cotton aphid is likely insensitive AChE (G. Moores personal communication) then control with both organophosphate and carbamate insecticides is probably at risk. Consecutive sprays of organophosphates or pirimicarb should be avoided. Pirimicarb can still be used in NSW but resistance has the potential to develop to an extreme level.

O'Brian *et al.* (1992), reported a significant loss of endosulfan and bifenthrin resistance, following seven months laboratory culture without insecticide exposure but found organophosphate resistance stable for up to eight months. Organophosphate and pirimicarb resistance stability in cotton aphid may limit the success of resistance management based on chemical alternation, because in the absence of immigration, alternation strategies rely upon fitness costs associated with resistance (Tabashnik 1990).

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